population utilizing 0.1% of the RfD. A Tier 3 chronic analysis was done using the DEEM™ software, Version 7.76 (Novigen Sciences, Inc.). The 1994–96, 1998 CSFII consumption data from USDA were used. Average anticipated residue values were calculated from the appropriate field trial studies conducted for pyrimethanil. The average residue values were adjusted by the projected PCT at product maturity. Concentration factors derived from processing studies were included where appropriate. Secondary residues were calculated using theoretical dietary burdens derived from sensible diets for beef and dairy cattle and tissue to feed ratios from the ruminant feeding study.

ii. *Drinking water*. U.S. EPA's Standard Operating Procedure (SOP) for Drinking Water Exposure and Risk Assessments was followed to perform the Tier 1 drinking water assessment. This SOP uses a variety of tools to conduct drinking water assessments, including water models such as Screening Concentration in Ground Water (SCI-GROW), FIRST, Pesticide Root Zone Model (PRZMS)/EXAMS, and monitoring data. If monitoring data are not available then the models are used to predict potential residues in surface and ground water and the highest levels (whether ground or surface) are assumed to be the drinking water residue. In the case of pyrimethanil, monitoring data are not available. SCI-GROW and FIRST were used to estimate a drinking water residue. Calculation of the Drinking Water Estimate Concentration (DWEC) for surface water for the worst case pyrimethanil use scenario results in an acute DWEC of 122 parts per billion (ppb) and a chronic DWEC of 37 ppb. DWLOCs calculated based on the acute and chronic risk assessments described above are many fold higher than these conservative DWECs. The adult acute and chronic DWLOCs are 10,146 ppb and 5,944 ppb respectively. Children's acute and chronic DWLOCs are 2,762 ppb and 1,695 ppb respectively.

2. *Non-dietary exposure*. Pyrimethanil products are not labeled for residential uses (food or non-food), thereby eliminating the potential for residential exposure or non-occupational exposure.

D. Cumulative Effects

Section 408(b)(2)(D)(v) requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider ''available information'' concerning the cumulative effects of a particular pesticide's residues and ''other substances that have a common mechanism of toxicity.'' There are no available data to determine whether pyrimethanil has a common mechanism of toxicity with other substances or how to include this pesticide in a cumulative risk assessment. Unlike other pesticides for which EPA has followed a cumulative risk approach based on a common mechanism of toxicity, pyrimethanil does not appear to form a toxic metabolite produced by other substances. For the purposes of the tolerance petition and this reduced risk rationale, therefore, it has been assumed that pyrimethanil does not have a common mechanism of toxicity with other substances.

E. Safety Determination

1. *U.S. population*. Using the assumptions and data described above, based on the completeness and reliability of the toxicity data, it is concluded that dietary risk from the proposed uses of pyrimethanil are acceptable for all populations examined. Chronic exposure for the U.S. population utilizes 0.1% (0.00015 mg/kg bwt/day) of the cRfD. Acute exposure for the U.S. population utilizes 3.4% (0.01012 mg/kg bwt/day) of the aRfD. The most highly exposed population of children 1–6 utilizes only 0.3% of the cRfD and 7.9% of the aRfD. The actual exposures are likely to be much less as more realistic data and models are developed. EPA generally has no concern for exposures below 100% of the RfD (acute or chronic), because the RfD represents the level at or below which exposure will not pose appreciable risk to human health. DWLOC for adults both acute (10,146 ppb) and chronic (5,944 ppb) are several orders of magnitude above the conservative DWEC for acute (122 ppb) and chronic (37 ppb) worst case scenarios. Therefore, there is a reasonable certainty that no harm will occur to the U.S. population from aggregate exposure (food and drinking water) to residues of pyrimethanil.

2. *Infants and children*. The relevant toxicity studies as discussed in the toxicology section above show no extra sensitivity of infants and children to pyrimethanil, therefore, the FQPA safety factor can be removed. Using the assumptions and data described in the exposure section above, it is concluded that dietary risk from the proposed uses of pyrimethanil are acceptable for all infant and children sub-populations examined. The most highly exposed sub-population was children 1–6 for both the chronic and acute analyses. The sub-population children 1–6 utilizes 0.3% (0.00047 mg/kg bwt/day) of the cRfD and 7.9% (0.02377 mg/kg bwt/day) of the aRfD. All other infant and children populations have less exposure. The chronic and acute drinking water levels of concern for children (1,695 ppb and 2,762 ppb respectively) are well above the conservative DWEC for chronic and acute scenarios. The chronic DWEC is 37 ppb and the acute DWEC is 122 ppb. Therefore, there is a reasonable certainty that no harm will occur to infants and children from aggregate exposure to residues of pyrimethanil.

F. International Tolerances

Maximum residue limits for pyrimethanil have not been established by the Codex Alimentarius Commission.

[FR Doc. 03–5032 Filed 3–4–03; 8:45 am]
BILLING CODE 6560–50–S

ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0027; FRL–7291–1]

Imidacloprid; Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket ID number OPP–2003–0027, must be received on or before April 4, 2003.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Dani Daniel, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–5409; e-mail address: *daniel.dani@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. Potentially affected entities may include, but are not limited to:

- Crop production (NAICS 111)
- Animal production (NAICS 112)
- Food manufacturing (NAICS 311)
- Pesticide manufacturing (NAICS 32532)

This listing is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in this unit could also be affected. The North American Industrial Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. If you have any questions regarding the applicability of this action to a particular entity, consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

B. How Can I Get Copies of this Document and Other Related Information?

1. *Docket*. EPA has established an official public docket for this action under docket identification (ID) number OPP–2003–0027. The official public docket consists of the documents specifically referenced in this action, any public comments received, and other information related to this action. Although a part of the official docket, the public docket does not include Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. The official public docket is the collection of materials that is available for public viewing at the Public Information and Records Integrity Branch (PIRIB), Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA. This docket facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The docket telephone number is (703) 305–5805.

2. *Electronic access*. You may access this **Federal Register** document electronically through the EPA Internet under the''**Federal Register**'' listings at http://www.epa.gov/fedrgstr/.

An electronic version of the public docket is available through EPA's electronic public docket and comment system, EPA Dockets. You may use EPA Dockets at http://www.epa.gov/edocket/ to submit or view public comments, access the index listing of the contents of the official public docket, and to access those documents in the public docket that are available electronically. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B.1. Once in the system, select ''search,'' then key in the appropriate docket ID number.

Certain types of information will not be placed in the EPA Dockets. Information claimed as CBI and other information whose disclosure is restricted by statute, which is not included in the official public docket, will not be available for public viewing in EPA's electronic public docket. EPA's policy is that copyrighted material will not be placed in EPA's electronic public docket but will be available only in printed, paper form in the official public docket. To the extent feasible, publicly available docket materials will be made available in EPA's electronic public docket. When a document is selected from the index list in EPA Dockets, the system will identify whether the document is available for viewing in EPA's electronic public docket. Although not all docket materials may be available electronically, you may still access any of the publicly available docket materials through the docket facility identified in Unit I.B. EPA intends to work towards providing electronic access to all of the publicly available docket materials through EPA's electronic public docket.

For public commenters, it is important to note that EPA's policy is that public comments, whether submitted electronically or in paper, will be made available for public viewing in EPA's electronic public docket as EPA receives them and without change, unless the comment contains copyrighted material, CBI, or other information whose disclosure is restricted by statute. When EPA identifies a comment containing copyrighted material, EPA will provide a reference to that material in the version of the comment that is placed in EPA's electronic public docket. The entire printed comment, including the copyrighted material, will be available in the public docket.

Public comments submitted on computer disks that are mailed or delivered to the docket will be transferred to EPA's electronic public docket. Public comments that are mailed or delivered to the docket will be scanned and placed in EPA's electronic public docket. Where practical, physical objects will be photographed, and the photograph will be placed in EPA's electronic public docket along with a brief description written by the docket staff.

C. How and To Whom Do I Submit Comments?

You may submit comments electronically, by mail, or through hand delivery/courier. To ensure proper receipt by EPA, identify the appropriate docket ID number in the subject line on the first page of your comment. Please ensure that your comments are submitted within the specified comment period. Comments received after the close of the comment period will be marked ''late.'' EPA is not required to consider these late comments. If you wish to submit CBI or information that is otherwise protected by statute, please follow the instructions in Unit I.D. Do not use EPA Dockets or e-mail to submit CBI or information protected by statute.

1. *Electronically*. If you submit an electronic comment as prescribed in this unit, EPA recommends that you include your name, mailing address, and an e-mail address or other contact information in the body of your comment. Also include this contact information on the outside of any disk or CD ROM you submit, and in any cover letter accompanying the disk or CD ROM. This ensures that you can be identified as the submitter of the comment and allows EPA to contact you in case EPA cannot read your comment due to technical difficulties or needs further information on the substance of your comment. EPA's policy is that EPA will not edit your comment, and any identifying or contact information provided in the body of a comment will be included as part of the comment that is placed in the official public docket, and made available in EPA's electronic public docket. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment.

i. *EPA Dockets*. Your use of EPA's electronic public docket to submit comments to EPA electronically is EPA's preferred method for receiving comments. Go directly to EPA Dockets at http://www.epa.gov/edocket, and follow the online instructions for submitting comments. Once in the system, select ''search,'' and then key in docket ID number OPP–2003–0027 The system is an ''anonymous access'' system, which means EPA will not know your identity, e-mail address, or other contact information unless you provide it in the body of your comment.

ii. *E-mail*. Comments may be sent by e-mail to opp-docket@epa.gov, Attention: Docket ID number OPP–2003–0027. In contrast to EPA's electronic public docket, EPA's e-mail system is not an ''anonymous access'' system. If you send an e-mail comment directly to the docket without going through EPA's electronic public docket, EPA's e-mail system automatically captures your e-mail address. E-mail addresses that are automatically captured by EPA's e-mail system are included as part of the comment that is

placed in the official public docket, and made available in EPA's electronic public docket.

iii. *Disk or CD ROM.* You may submit comments on a disk or CD ROM that you mail to the mailing address identified in Unit I.C.2. These electronic submissions will be accepted in WordPerfect or ASCII file format. Avoid the use of special characters and any form of encryption.

2. *By mail.* Send your comments to: Public Information and Records Integrity Branch (PIRIB) (7502C), Office of Pesticide Programs (OPP), Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001, Attention: Docket ID number OPP–2003–0027.

3. *By hand delivery or courier.* Deliver your comments to: Public Information and Records Integrity Branch (PIRIB), Office of Pesticide Programs (OPP), Environmental Protection Agency, Rm. 119, Crystal Mall #2, 1921 Jefferson Davis Hwy., Arlington, VA, Attention: Docket ID number OPP–2003–0027. Such deliveries are only accepted during the docket's normal hours of operation as identified in Unit I.B.1.

D. How Should I Submit CBI To the Agency?

Do not submit information that you consider to be CBI electronically through EPA's electronic public docket or by e-mail. You may claim information that you submit to EPA as CBI by marking any part or all of that information as CBI (if you submit CBI on disk or CD ROM, mark the outside of the disk or CD ROM as CBI and then identify electronically within the disk or CD ROM the specific information that is CBI). Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

In addition to one complete version of the comment that includes any information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket and EPA's electronic public docket. If you submit the copy that does not contain CBI on disk or CD ROM, mark the outside of the disk or CD ROM clearly that it does not contain CBI. Information not marked as CBI will be included in the public docket and EPA's electronic public docket without prior notice. If you have any questions about CBI or the procedures for claiming CBI, please consult the person listed under **FOR FURTHER INFORMATION CONTACT**.

E. What Should I Consider as I Prepare My Comments for EPA?

You may find the following suggestions helpful for preparing your comments:

1. Explain your views as clearly as possible.

2. Describe any assumptions that you used.

3. Provide copies of any technical information and/or data you used that support your views.

4. If you estimate potential burden or costs, explain how you arrived at the estimate that you provide.

5. Provide specific examples to illustrate your concerns.

6. Make sure to submit your comments by the deadline in this notice.

7. To ensure proper receipt by EPA, be sure to identify the docket ID number assigned to this action in the subject line on the first page of your response. You may also provide the name, date, and **Federal Register** citation.

II. What Action is the Agency Taking?

EPA has received a pesticide petition as follows proposing the establishment and/or amendment of regulations for residues of a certain pesticide chemical in or on various food commodities under section 408 of the Federal Food, Drug, and Cosmetic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that this petition contains data or information regarding the elements set forth in FFDCA section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data support granting of the petition. Additional data may be needed before EPA rules on the petition.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: February 11, 2003.

Debra Edwards,

Acting Director, Registration Division, Office of Pesticide Programs.

Summary of Petition

The petitioner's summary of the pesticide petition is printed below as required by FFDCA section 408(d)(3). The summary of the petition was prepared by the petitioner and represents the views of the petitioner. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

Bayer CropScience

PP 0E6074

EPA has received a pesticide petition (0E6074) from Bayer CropScience, 2 T.W. Alexander Drive, PO Box 12014, Research Triangle Park, NC 27709 proposing, pursuant to section 408(d) of the FFDCA, 21 U.S.C. 346a(d), to amend 40 CFR 180.472, by establishing an import tolerance for residues of imidacloprid, [(1-[6-chloro-3-pyridinyl) methyl]-*N*-nitro-2-imidazolidinimine) and its metabolites containing the 6-chloropyridinyl moiety, all expressed as 1-[(6-chloro-3-pyridinyl)methyl]-*N*-nitro-2-imidazolidinimine] in or on the raw agricultural commodity (RAC): Banana at 0.01 parts per million (ppm). EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism.* The nature of the imidacloprid residue in plants and livestock is adequately understood. The residues of concern are combined residues of imidacloprid and it metabolites containing the 6-chloropyridinyl moiety, all calculated as imidacloprid.

2. *Analytical method.* The analytical method is a common moiety method for imidacloprid and its metabolites containing the 6-chloropyridinyl moiety using a permanganate oxidation, silyl derivatization, and capillary gas chromatography mass spectrometry (GC-MS) selective ion monitoring. This method has successfully passed a petition method validation in EPA labs. There is a confirmatory method specifically for imidacloprid and several metabolites utilizing GC/MS and high performance liquid chromatography ultraviolet (HPLC-UV) which has been validated by EPA as well. Imidacloprid and its metabolites are stable for at least 24 months in the commodities when frozen.

3. *Magnitude of residues.* For bananas, Bayer conducted 12 residue crop field trials to evaluate the quantity of imidacloprid expected in bananas from applications of Confidor 70 WG and Confidor 350 SC. Trials were conducted at eight sites in the Caribbean coastal area of Central America and 4 sites in

the Pacific coastal area of Ecuador. Imidacloprid residues in banana whole fruit were quantitated by GC using a mass selective detector. The limit of quantitation (LOQ) was 0.01 ppm. The highest average field trial (HAFT) was 0.01 ppm in bananas.

B. Toxicological Profile

1. *Acute toxicity*. The acute oral LD_{50} values for imidacloprid technical ranged from 424 milligrams/kilograms (mg/kg) in the male rat and <450 mg/kg in the female rat. The acute dermal LD_{50} was <5,000 mg/kg in the rat. The 4–hour rat inhalation LC_{50} was <5.33 mg/L. Imidacloprid was not irritating to rabbit skin or eyes. Imidacloprid did not cause skin sensitization in guinea pigs. In an acute neurotoxicity study the lowest observed adverse effect level (LOAEL) = 42 mg/kg body weight day (bwt/day).

2. *Genotoxicty*. Mutagenicity studies have demonstrated that imidacloprid is non-mutagenic both *in vivo* and *in vitro*.

3. *Reproductive and developmental toxicity*. In a developmental toxicity study with Sprague-Dawley rats, groups of pregnant animals (25/group) received oral administration of imidacloprid (94.2%) at 0, 10, 30, or 100 mg/kg bwt/day during gestation days 6 through 16. Maternal toxicity was manifested as decreased body weight gain at all dose levels and reduced food consumption at 100 milligrams/kilograms of body weight/day (mg/kg bwt/day. No treatment-related effects were seen in any of the reproductive parameters (i.e., Cesarean section evaluation). At 100 mg/kg bwt/day, developmental toxicity manifested as wavy ribs (fetus = 7/149 in treated vs. 2/158 in controls and litters, 4/25 vs. 1/25). For maternal toxicity, the LOAEL was 10 mg/kg bwt/day lowest dose tested (LDT) based on decreased body weight gain; a NOAEL was not established. For developmental toxicity, the NOAEL was 30 mg/kg bwt/day and the LOAEL was 100 mg/kg bwt/day based on increased wavy ribs master record identification (MRID No. 42256338). In a developmental toxicity study with Chinchilla rabbits, groups of 16 pregnant does were given oral doses of imidacloprid (94.2%) at 0, 8, 24, or 72 mg/kg bwt/day during gestation days 6 through 18. For maternal toxicity, the NOAEL was 24 mg/kg bwt/day and the LOAEL was 72 mg/kg bwt/day based on mortality, decreased body weight gain, increased resorptions, and increased abortions. For developmental toxicity, the NOAEL was 24 mg/kg bwt/day and the LOAEL was 72 mg/kg bwt/day based on decreased fetal body weight, increased resorptions, and increased skeletal abnormalities (MRID No. 42256339). In a 2-generation reproductive toxicity study, imidacloprid (95.3%) was administered to Wistar/Han rats at dietary levels of 0, 100, 250, or 700 ppm (0, 7.3, 18.3, or 52.0 mg/kg bwt/day for males and 0, 8.0, 20.5, or 57.4 mg/kg bwt/day for females) (MRID No. 42256340, Doc. No. 010537). For parental/systemic/reproductive toxicity, the NOAEL was 250 ppm (18.3 mg/kg bwt/day) and the LOAEL was 750 ppm (52 mg/kg bwt/day), based on decreases in body weight in both sexes in both generations. Based on these factors, EPA recommended that the Data Evaluation Record should be revised to indicate the parental/systemic/reproductive NOAEL and LOAEL to be 250 and 700 ppm, respectively, based upon the body weight decrements observed in both sexes in both generations.

4. *Subchronic toxicity*. In a dermal toxicity study, groups of five male and five female New Zealand white rabbits received repeated dermal applications of imidacloprid (95%) at 1,000 mg/kg bwt/day (LD), 6 hours/day, 5 days/week for 3 weeks. No dermal or systemic toxicity was seen. For systemic and dermal toxicity, the NOAEL was <1,000 mg/kg bwt/day, a LOAEL was not established (MRID No. 42256329). In an oral toxicity study, groups of Fischer 344 rats (12/sex/dose) were fed diets containing imidacloprid (98.8%) at 0, 150, 1,000, or 3,000 ppm (0, 9.3, 63.3, or 196 mg/kg bwt/day in males and 0, 10.5, 69.3 or 213 mg/kg bwt/day in females, respectively) for 90 days. No treatment-related effects were seen at 150 ppm. Treatment-related effects included decreases in body weight gain during the first 4 weeks of the study at 1,000 ppm (22% in males and 18% in females) and 3,000 ppm (50% in males and 25% in females) with an associated decrease in forelimb grip strength especially in males. The NOAEL was 150 ppm (9.3 and 10.5 mg/kg bwt/day in males and females, respectively) and the LOAEL was 1,000 ppm (63.3 and 69.3 mg/kg bwt/day in males and females, respectively) (MRID No. 43286401). In a rat inhalation study (28–day study in which rats were exposed 6 hours/day, 5 days/week for 4 weeks), the NOAEL for imidacloprid was 5.5 mg/m^3 (MRID No. 422730–01).

5. *Chronic toxicity*. In a chronic toxicity study, groups of beagle dogs (4/sex/dose) were fed diets containing imidacloprid (94.9%) at 0, 200 or 1,250/2,500 ppm (0, 6.1, 15 or 41/72 mg/kg bwt/day, respectively) for 52 weeks. The 1,250 ppm dose was increased to 2,500 ppm from week 17 onwards. The threshold NOAEL was 1,250 ppm (41 mg/kg bwt/day). The LOAEL was 2,500 ppm (72 mg/kg bwt/day) based on increased cytochrome-P-450 levels in both sexes and was considered to be a threshold dose. Due to the lack of toxicity at 1,250 ppm, a LOAEL was not established in this study following the dose increase to the 2,500 ppm level, toxicity was observed, thus making 1,250 ppm the threshold NOAEL and 2,500 ppm the threshold LOAEL (MRID No. 42273002).

6. *Animal metabolism*. The metabolism imidacloprid in rats was reported in seven studies. These data show that imidacloprid was rapidly absorbed and eliminated in the excreta (90% of the dose within 24 hours), demonstrating no biologically significant differences between sexes, dose levels, or route of administration. Elimination was mainly renal (70–80% of the dose) and fecal (17–25%). The major part of the fecal activity originated in the bile. Total body accumulation after 48 hours consisted of 0.5% of the radioactivity with the liver, kidney, lung, skin and plasma being the major sites of accumulation. Therefore, bioaccumulation of imidacloprid is low in rats. Maximum plasma concentration was reached between 1.1 and 2.5 hours. Two major routes of biotransformation were proposed for imidacloprid. The first route included an oxidative cleavage of the parent compound rendering 6-chloronicotinic acid and its glycine conjugate. Dechlorination of this metabolite formed the 6-hydroxynicotinic acid and its mercapturic acid derivative. The second route included the hydroxylation followed by elimination of water of the parent compound rendering imidacloprid. A comparison between [methylene-^{14}C]-imidacloprid and [imidazolidine-4,5-^{14}C]-imidacloprid showed that while the rate of excretion was similar, the renal portion was higher with the imidazolidine-labeled compound. In addition, accumulation in tissues was generally higher with the imidazolidine-labeled compound. A comparison between imidacloprid and one of its metabolites, WAK 3839, showed that the total elimination was the same for both compounds. The proposed metabolic pathways for these two compounds were different. WAK 3839 was formed following pretreatment (repeated dosing) of imidacloprid.

7. *Endocrine disruption*. The toxicology data base for imidacloprid is current and complete. Studies in this data base include evaluation of the potential effects on reproduction and development, and an evaluation of the pathology of the endocrine organs following short-term or long-term exposure. These studies revealed no

primary endocrine effects due to imidacloprid.

C. Aggregate Exposure

1. *Dietary exposure*. Assessments were conducted to evaluate potential risks due to chronic and acute dietary exposure of the U.S. population and selected population subgroups to residues of imidacloprid. These analyses cover all registered crops including rotational crops; uses pending with EPA in the 2003 work plan including dry beans, peas, bushberries, lingonberry, juneberries, salal, carrots, turnips, okra, cranberries, artichoke (globe), watercress, beet roots, leaves of root and tuber vegetables, stone fruit, mamey sapote, guava, feijoa, jaboticaba, wax jambu, starfruit, passion fruit, acerola, strawberry, cucumber (greenhouse), and tomato (greenhouse), this import tolerance petition on bananas; and active and proposed section 18 uses on blueberries, cranberries, table beets, strawberries, turnips.

Novigen Sciences, Inc.'s Dietary Exposure Evaluation Model (DEEM™), which is licensed to Bayer, was used to estimate the chronic and acute dietary exposure. This software uses the food consumption data from the 1994–1998 Department of Agriculture (USDA) continuing surveys of food intake by individuals (CSFII) 1994–1998.

The endpoint for acute dietary risk assessments is based on neurotoxicity characterized by decreases in motor or locomotor activity in female rats at 42 mg/kg bwt/day, the LOAEL from an acute neurotoxicity study. Based on an uncertainty factor (UF) of 10x for inter-species and 10x for intra-species the acute reference dose (aRfD) = 0.42 mg/kg bwt/day. EPA has determined that an additional UF for FQPA (reduced to 3x) applies to all population subgroups for acute risk. Application of the additional 3x safety factor results in an acute population adjusted dose (aPAD) 0.14 mg/kg bwt/day or a margin of exposure (MOE) of 300. For chronic dietary analyses, EPA has established the reference dose (RfD) for imidacloprid at 0.057 mg/kg/day based on a NOAEL of 5.7 mg/kg bwt/day from a rat chronic toxicity carcinogenicity study and uncertainty factors of 10x for inter-species and 10x for intra-species. EPA has determined that an additional UF for FQPA (reduced to 3x) applies to all population subgroups for chronic risk. Application of the additional 3x safety factor results in a chronic population adjusted dose (cPAD) of 0.019 mg/kg bwt/day. Results from the acute and chronic dietary exposure analyses described below demonstrate a reasonable certainty that no harm to the overall U.S. population or any population subgroup will result from the use of imidacloprid on currently registered and pending uses.

i. *Food*. Acute and chronic (tier 3) risk assessments were made using the results of field trials conducted at maximum label application rates and the shortest pre-harvest intervals. For some of the vegetable crops, these residue data were collected at 1.5x or greater than the maximum label rate of 0.5 lb active ingredient/acre per season. In addition, no adjustments were made to account for dissipation of residues during storage, transportation from the field to the consumer, washing or peeling. Therefore, the actual dietary exposure will be less than that presented here. For the chronic analysis, mean field trial residues were calculated. For the acute Monte Carlo analysis, the entire distribution of residue field trial data was used for the ''non-blended'' and ''partially blended'' foods as determined by EPA's HED SOP 99.6. For the foods considered as ''blended'' by EPA'S HED SOP 99.6, mean field trial residue data were used. As allowed in EPA's draft guidance for submission of probabilistic human health exposure assessments one half limit of detection (LOD)/LOQ values were used for all non-detected values (values below the sensitivity of the method). Bayer's acute Monte Carlo dietary exposure assessment estimated percent of the aPAD and corresponding MOE for the overall U.S. population, (all seasons), and various subpopulations. In this analysis, the exposure for the total U.S. population was equal to 7.73% of the aPAD at the 99.9th percentile. The most highly exposed population subgroup, children (1–6 years), had an exposure equal to 16.42% of the aPAD at the 99.9th percentile. Therefore, the acute dietary exposure estimates are below EPA's level of concern for the overall U.S. population as well as the various subpopulations. Bayer's chronic dietary exposure estimated the percent of the cPAD for the overall U.S. population (all seasons) and various subpopulations. In this analysis, the exposure for the total U.S. population was equal to 1.4% of the cPAD. The most highly exposed population subgroup, children (1-6 years), had an exposure equal to 3.0% of the cPAD. Therefore, the chronic exposure estimates are below EPA's level of concern for the overall U.S. population as well as the various subpopulations.

ii. *Drinking water*. EPA, as published in the **Federal Register** of April 10, 2001 (66 FR 18554) (FRL–6777–6), calculated acute and chronic DWLOCs and compared them with the EECs for surface and ground water. Based on this comparison, they determined that acute exposure and chronic exposure would not be expected to exceed the aPAD and cPAD, respectively. It is not expected that the additional exposure from the minor crops pending in EPA's 2003 work plan would significantly change EPA's water assessment.

2. *Non-dietary exposure*—i. *Residential turf*. Bayer has conducted an exposure study to address the potential exposures of adults and children from contact with imidacloprid treated turf. The population considered to have the greatest potential exposure from contact with pesticide treated turf soon after pesticides are applied are young children. Margins of safety (MOS) of 7,587 - 41,546 for 10–year old children and 6,859 - 45,249 for 5–year old children were estimated by comparing dermal exposure doses to the imidacloprid NOAEL of 1,000 mg/kg/day established in a 15–day dermal toxicity study in rabbits. The estimated safe residue levels of imidacloprid on treated turf for 10–year old children ranged from 5.6 - 38.2 μg/cm^2 and for 5–year old children from 5.1 - 33.5 μg/cm^2. This compares with the average imidacloprid transferable residue level of 0.080 μg/cm^2 present immediately after the sprays have dried. These data indicate that children can safely contact imidacloprid-treated turf as soon after application as the spray has dried.

ii. *Termiticide*. Imidacloprid is registered as a termiticide. Due to the nature of the treatment for termites, exposure would be limited to that from inhalation and was evaluated by EPA and Bayer. Data indicate that the MOS for the worst case exposures for adults and infants occupying a treated building who are exposed continuously (24 hours/day) are 8.0 x 10^{-7} and 2.4 x 10^{-8}, respectively - and exposure can thus be considered negligible.

iii. *Tobacco*. Smoke Studies have been conducted to determine residues in tobacco and the resulting smoke following treatment. Residues of imidacloprid in cured tobacco following treatment were a maximum of 31 ppm (7 ppm in fresh leaves). When this tobacco was burned in a pyrolysis study only 2% of the initial residue was recovered in the resulting smoke (main stream plus side stream). This would result in an inhalation exposure to imidacloprid from smoking of approximately 0.0005 mg per cigarette. Using the measured subacute rat inhalation NOAEL of 5.5 mg/m^3, it is apparent that exposure to imidacloprid from smoking (direct and/or indirect exposure) would not be significant.

iv. *Pet treatment.* Human exposure from the use of imidacloprid to treat dogs and cats for fleas has been addressed by EPA with the conclusion that due to the fact that imidacloprid is not an inhalation or dermal toxicant and that while dermal absorption data are not available, imidacloprid is not considered to present a hazard via the dermal route.

D. Cumulative Effects

Imidacloprid is a chloronicotinyl insecticide. At this time, EPA has not made a determination that imidacloprid and other substances that may have a common mechanism of toxicity would have cumulative effects. Therefore, for these tolerance petitions, it is assumed that imidacloprid does not have a common mechanism of toxicity with other substances and only the potential risks of imidacloprid in its aggregate exposure are considered.

E. Safety Determination

1. *U.S. population.* EPA has considered data from developmental toxicity studies in the rat and rabbit and a 2-generation reproduction study in the rat. These studies are discussed under section A (Toxicology Profile) above. The developmental toxicity data demonstrated no increased sensitivity of rats or rabbits to *in utero* exposure to imidacloprid. In addition, the multi-generation reproductive toxicity study did not identify any increased sensitivity of rats to *in utero* or post-natal exposure. Parental NOAELs were lower or equivalent to developmental or offspring NOAELs. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during gestation. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability of mating animals and data on systemic toxicity.

FFDCA section 408 provides that EPA shall apply an additional tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base unless EPA determines that a different MOS will be safe for infants and children. MOS are incorporated into EPA risk assessments either directly through use of a MOE analysis or through using uncertainty (safety) factors (UF) in calculating a dose level that poses no appreciable risk to humans. EPA believes that reliable data support using the standard UF (usually 100 for combined inter-species and intra-species variability) and not the additional tenfold MOE/UF when EPA has a complete data base under existing guidelines and when the severity of the effect in infants or children or the potency or unusual toxic properties of a compound do not raise concerns regarding the adequacy of the standard MOE/UF.

Although developmental toxicity studies showed no increased sensitivity in fetuses as compared to maternal animals following *in utero* exposures in rats and rabbits, no increased sensitivity in pups as compared to adults was seen in the 2-generation reproduction toxicity study in rats, and the toxicology data base is complete as to core requirements, EPA has determined that the additional safety factor for the protection of infants and children will be retained but reduced to 3x based on the following weight-of-the-evidence considerations relating to potential sensitivity and completeness of the data:

i. There is concern for structure activity relationship. Imidacloprid, a chloronicotinyl compound, is an analog to nicotine and studies in the published literature suggests that nicotine, when administered causes developmental toxicity, including functional deficits, in animals and/or humans that are exposed *in utero*.

ii. There is evidence that imidacloprid administration causes neurotoxicity following a single oral dose in the acute study and alterations in brain weight in rats in the 2–year carcinogenicity study.

iii. The concern for structure activity relationship along with the evidence of neurotoxicity dictates the need of a developmental neurotoxicity study for assessment of potential alterations on functional development. Because a developmental neurotoxicity study potentially relates to both acute and chronic effects in both the mother and the fetus, EPA has applied the additional UF for FQPA for all population subgroups, and in both acute and chronic risk assessments.

Based on the exposure assessments described above and on the completeness and reliability of the toxicity data, it can be concluded that the dietary exposure estimates from all label and pending uses of imidacloprid are 7.73% of the aPAD at the 99.9th percentile and 1.4% of the cPAD for the U.S. population. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

2. *Infants and children.* Based on the exposure assessments described above for the safety determination of the U.S. population and on the completeness and reliability of the toxicity data, it can be concluded that the dietary exposure estimates from all label and pending uses of imidacloprid are 16.42% of the aPAD at the 99.9th percentile and 3.0% of the cPAD for the most sensitive population subgroup, children 1–6 years. Thus, it can be concluded that there is a reasonable certainty that no harm will result from aggregate exposure to imidacloprid residues.

F. International Tolerances

No CODEX Maximum Residue Levels have been established for residues of imidacloprid on any crops pending in EPA's 2003 work plan.

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ENVIRONMENTAL PROTECTION AGENCY

[OPP–2003–0047; FRL–7294–5]

Trifloxystrobin; Notice of Filing a Pesticide Petition to Establish a Tolerance for a Certain Pesticide Chemical in or on Food

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of a pesticide petition proposing the establishment of regulations for residues of a certain pesticide chemical in or on various food commodities.

DATES: Comments, identified by docket ID number OPP–2003–0047, must be received on or before April 4, 2003.

ADDRESSES: Comments may be submitted electronically, by mail, or through hand delivery/courier. Follow the detailed instructions as provided in Unit I. of the **SUPPLEMENTARY INFORMATION**.

FOR FURTHER INFORMATION CONTACT: Sidney Jackson, Registration Division (7505C), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–7610; e-mail address: *jackson.sidney@epa.gov*.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. Potentially affected entities may include, but are not limited to: